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# Multiple Monomorphic Ventricular Tachycardia Configurations Predict Failure of Antiarrhythmic Drug Therapy Guided by Electrophysiologic Study

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***Objectives.*** **The purpose of this study was to determine whether the induction at electrophysiologic study of sustained monomorphic ventricular tachycardias with multiple QRS complex configurations predicted failure of subsequent serial electrophysiologic study guided antiarrhythmic drug testing.**

***Background.*** **Ventricular tachycardias with multiple QRS complex configurations are associated with failure of surgical therapy for ventricular tachycardia. As such, the presence of multiple monomorphic QRS complex ventricular tachycardias during electrophysiologic testing may predict failure of subsequent medical therapy.**

***Methods.*** **Fifty-one consecutive patients with coronary artery disease had reproducible induction of monomorphic ventricular tachycardia during a baseline electrophysiologic study. Each patient then underwent a mean of 1.5 antiarrhythmic drug trials. An antiarrhythmic drug regimen that suppressed induction of ventricular tachycardia was identified in 13 (26%) of the 51 patients.**

***Results.*** **Patients with only one inducible monomorphic QRS complex ventricular tachycardia at baseline study were more likely to have an antiarrhythmic drug regimen identified that suppressed inducible ventricular tachycardia than were patients with multiple monomorphic QRS complex ventricular tachycardias (12 [36%] of 33 patients vs. 1 [6%] of 18, p = 0.04). In seven patients with only one induced configuration of ventricular tachycardia, a second monomorphic ventricular tachycardia with a different QRS complex configuration occurred during attempts at pacing termination of the induced ventricular tachycardia. None of these seven patients then had successful drug suppression of inducible ventricular tachycardia. Thus, 12 (46%) of 26 patients with a single monomorphic QRS complex ventricular tachycardia observed at baseline study had successful serial drug testing compared with 1 (4%) of 25 patients with multiple QRS complex ventricular tachycardia configurations (p = 0.002).**

***Conclusions.*** **The induction or observation of multiple monomorphic QRS complex ventricular tachycardias at baseline electrophysiologic study predicted failure of subsequent serial electrophysiologic study–guided antiarrhythmic drug therapy.**

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Serial electrophysiologic study–guided drug testing has been used to direct therapy in patients with ventricular tachycardia. When antiarrhythmic drugs are identified that suppress ventricular tachycardia, the outcome is favorable (1–3). The likelihood of identifying a successful antiarrhythmic drug regimen that can suppress ventricular tachycardia induction has ranged from 25% to 70% (4–9). Thus, many patients who have unsuccessful serial electrophysiologic drug testing must be considered for alternative therapies (10).

Serial electrophysiologic drug testing is invasive, time-consuming and costly. If factors that predict failure of serial electrophysiologic drug testing were identified early, patient care would be optimized.

Various clinical and electrophysiologic factors have been examined to determine predictors of success or failure at serial electrophysiologic drug testing (5–8). Several studies (5,11,12) have reported that the response to intravenous procainamide at the initial electrophysiologic study predicted the outcome of electrophysiologic antiarrhythmic drug studies. However, other studies (13,14) have reported otherwise.

In the surgical treatment of ventricular tachycardia, the presence of more than one monomorphic ventricular tachycardia QRS complex configuration predicted failure of surgical treatment of ventricular tachycardia (15–17). Morphologically distinct monomorphic QRS complex ventricular

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tachycardias often originate at different left ventricular sites (16–19). We hypothesized that the presence of more than one QRS complex ventricular tachycardia configuration at electrophysiologic study would also predict failure of serial electrophysiologic study guided medical therapy.

## Methods

**Study patients.** The study group consisted of 51 consecutive patients with coronary artery disease who had a sustained monomorphic ventricular tachycardia that was reproducibly induced at the baseline study in the absence of antiarrhythmic drugs. These patients were selected from a larger pool of 84 patients who had serial drug testing for sustained monomorphic ventricular tachycardia. However, 11 patients without coronary artery disease were excluded, and 22 patients who did not have reinduction of ventricular tachycardia owing to hemodynamic instability during ventricular tachycardia were also excluded.

There were 44 men and 7 women, with a mean age of 66 years (range 41 to 82). All patients had coronary artery disease, as documented by angiographic data. The mean left ventricular ejection fraction was 30% (range 9% to 60%). Indications for electrophysiologic study included cardiac arrest in 5 patients, syncope in 11 and documented sustained ventricular tachycardia in 35.

**Electrophysiologic study.** After giving written informed consent, all patients had a baseline electrophysiologic study in the postabsorptive state. All antiarrhythmic drugs, excluding beta-adrenergic blocking agents and digoxin, were discontinued for at least five half-lives before the baseline study. Using standard techniques, multipolar electrode catheters were positioned in the high right atrium, across the tricuspid valve to record a His bundle electrogram and at the right ventricular apex or outflow tract, or both, for recording and stimulation. A programmable, battery-powered stimulator (Medtronic 5328) was used to deliver square-wave stimuli of 2.0-ms duration at twice the diastolic threshold. Surface electrocardiographic (ECG) leads I, II, aVF, $V_1$ and bipolar intracardiac electrograms were monitored and recorded on a multichannel oscilloscope (Electronics-for-Medicine VR16 recorder), and hard copy tracings were obtained at paper speeds of 50 to 100 mm/s. All data were recorded simultaneously on FM magnetic tape (Honeywell model 101).

A standard programmed ventricular stimulation protocol was used (20). The programmed stimulation protocol included introduction of single, double and triple ventricular extrastimuli after an 8-beat drive train at cycle lengths of 600, 500, or 400 ms. Ventricular extrastimuli were introduced to scan diastole in decrements of 10 ms until ventricular tachycardia was induced or ventricular refractoriness was achieved at two right ventricular sites. Sustained ventricular tachycardia was reinduced at baseline study in the absence of antiarrhythmic drug therapy.

**Antiarrhythmic drug trials.** Serial drug testing was performed, with only suppression of induction of ventricular tachycardia as a positive end point. An intravenous type IA antiarrhythmic drug was usually tested during the initial setting after completion of the baseline study. Subsequent electrophysiologic drug trials utilized orally administered class IA, IB, IC or III antiarrhythmic drugs. Electrophysiologic studies performed utilizing amiodarone were excluded from this report.

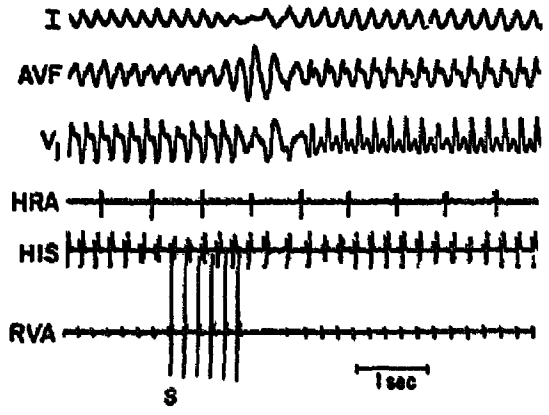


**Figure 1.** Electrocardiographic leads I, aVF and $V_1$ recorded simultaneously with electrograms from the high right atrium (HRA), His bundle (HIS) and right ventricular apex (RVA) during ventricular tachycardia and during a single pacing attempt to interrupt the ventricular tachycardia. Note that after the introduction of the pacing train from the right ventricular apex, a monomorphic ventricular tachycardia with a different QRS complex configuration than that of the initially induced ventricular tachycardia developed. S = stimulus artifact.

**Ventricular tachycardia QRS complex configurations.** In each patient, the 12-lead ECGs of all monomorphic ventricular tachycardia QRS complex configurations were compared with each other. Electrocardiographic electrodes were placed in the standard position during the baseline study. In defining monomorphic ventricular tachycardia QRS complexes as similar, the bundle branch block pattern in lead $V_1$, mean frontal axis to within 30° and the R wave progression across the precordium were identical. In addition to comparing the QRS configurations of all induced sustained ventricular tachycardias, we also compared the QRS complex configurations of all observed sustained monomorphic ventricular tachycardias (i.e., those that resulted from induction at programmed stimulation or from attempted pacing termination) (Fig. 1). The data analysis was performed for 1) ventricular tachycardias that were induced by programmed stimulation, and 2) ventricular tachycardias that were induced or resulted from attempted pacing termination.

Twenty-five patients underwent a drug trial using intravenous procainamide (15 mg/kg body weight) after completion of the baseline study. We compared the QRS complex configurations of all sustained ventricular tachycardias (either induced or resulting from antitachycardia pacing) after intravenous procainamide relative to the monomorphic ventricular tachycardia QRS complex configurations observed during the baseline study.

Each patient's spontaneous (clinical) ventricular tachycardia was not included in the analysis. At electrophysiologic study, polymorphic ventricular tachycardia and ventricular fibrillation were excluded from analysis in this study.

**Data analysis.** Unpaired data were compared by the two-tailed *t* test for continuous variables and by chi-square analysis or the Fisher exact test for comparisons of proportions. In addition, a stepwise multiple logistic regression analysis was performed to determine whether any of the following variables correlated with successful electrophysiologic study guided drug therapy: age, gender, ejection fraction, minimal number of extrastimuli required to induce ventricular tachycardia at baseline study, induced ventricular tachycardia cycle length, number of ventricular tachycardia QRS complex configurations at baseline study and clinical presentation.

**Definition of terms.** Standard definitions were used for sustained ventricular tachycardia, nonsustained ventricular tachycardia, monomorphic ventricular tachycardia, multiple monomorphic ventricular tachycardias and polymorphic ventricular tachycardia (20). *Successful electrophysiologic study guided therapy* was defined as completion of the ventricular stimulation protocol in the presence of antiarrhythmic drug therapy without inducing sustained ventricular tachycardia. *Positive predictive value* was defined as the number of patients with one ventricular tachycardia QRS complex configuration who had successful electrophysiologic study–guided therapy, divided by all patients with one ventricular tachycardia QRS complex configuration. *Negative predictive value* was defined as the number of patients with multiple monomorphic QRS complex ventricular tachycardias who did not have successful electrophysiologic study–guided therapy, divided by all patients with multiple monomorphic ventricular tachycardias. *Baseline study* was defined as the electrophysiologic study performed in the absence of antiarrhythmic drugs.

## Results

**Electrophysiologic profile.** The mean induced tachycardia cycle length was 290 ms. The mean number of ventricular extrastimuli required to induce ventricular tachycardia at baseline electrophysiologic study was 2.2 ± 0.7. Patients underwent a mean of 1.5 antiarrhythmic drug trials.

Overall, 13 (26%) of 51 patients had successful electrophysiologic study–guided therapy (Table 1). These 13 patients underwent a mean of 1.4 antiarrhythmic drug trials, whereas the 38 patients without successful electrophysiologic study–guided drug therapy underwent a mean of 1.6 antiarrhythmic drug trials (p = NS). A variety of drugs or drug combinations were used (Table 2).

**Induction of multiple monomorphic sustained ventricular tachycardias.** Eighteen (36%) of 51 patients had induction of multiple monomorphic ventricular tachycardias at the baseline study (Fig. 1). Only 1 (6%) of these 18 patients subsequently had successful electrophysiologic study–guided medical therapy. However, 12 (36%) of the 33 patients with reproducible induction of morphologically similar ventricular tachycardias at the baseline electrophysiologic study had successful electrophysiologic study–guided medical therapy (Fig. 2) (p = 0.04).

Table 1. Outcome of Electrophysiologic Study in 51 Patients With Coronary Artery Disease

| | VT Suppressed (n = 13) | VT Not Suppressed (n = 38) |
|---|---|---|
| Induced VT cycle length | 296 ± 16 | 284 ± 8 |
| Extrastimuli used to induce VT at baseline EPS (no.) | | |
| 0 | 0 | 1 |
| 1 | 2 | 4 |
| 2 | 7 | 20 |
| 3 | 4 | 13 |
| EPS drug trials | | |
| 1 | 8 | 18 |
| 2 | 5 | 18 |
| 3 | 0 | 2 |

Values presented are mean value ± SD or number. EPS = electrophysiologic study; VT = ventricular tachycardia.

Of the 33 patients with reproducible induction of monomorphic ventricular tachycardia with similar QRS complex configurations, 7 subsequently developed a monomorphic ventricular tachycardia with a different QRS complex configuration during attempted pacing termination. These seven patients subsequently did not have successful electrophysiologic study–guided medical therapy. Thus, only 1 (4%) of 25 patients with multiple ventricular tachycardia QRS complex configurations observed at baseline study had successful electrophysiologic drug suppression in contrast to 12 (46%) of 26 patients with only one observed ventricular tachycardia QRS complex configuration (Fig. 3) (p = 0.002).

The outcome of electrophysiologic drug testing after each of the first two drug trials is shown in Figure 4. After baseline study, the outcome of the first drug trial was successful in 9 of 26 patients with a single monomorphic ventricular tachycardia QRS complex configuration compared with 1 of 25 patients with multiple monomorphic ventricular tachycardia QRS complex configurations (p = 0.016). After the first drug trial, 23 patients who had an unsuccessful first drug trial

Table 2. Drugs Used During Serial Electrophysiologic Study

| | Patients (no.) |
|---|---|
| Procainamide | |
| Intravenous | 25 |
| Oral | 1 |
| Quinidine (oral) | 12 |
| Quinidine/mexiletine (oral) | 29 |
| Type IC (encainide or flecainide) (oral) | 6 |
| Sotalol (oral) | 2 |
| Lidocaine (intravenous) | 2 |
| Encainide/mexiletine (oral) | 1 |

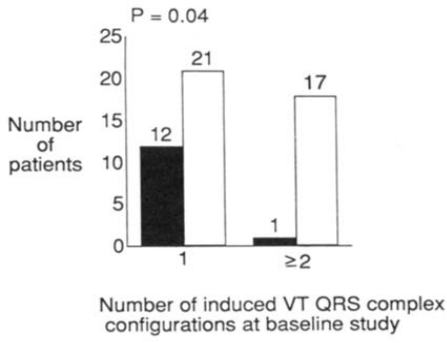


**Figure 2.** Histogram demonstrating outcome of electrophysiologic study-guided antiarrhythmic drug therapy based on the number of ventricular tachycardia (VT) QRS complex configurations induced at baseline study. The difference between successful therapy in the two groups (1 vs. ≥2) was statistically significant. **Black bars** = successful therapy; **white bars** = unsuccessful therapy. See text for discussion.

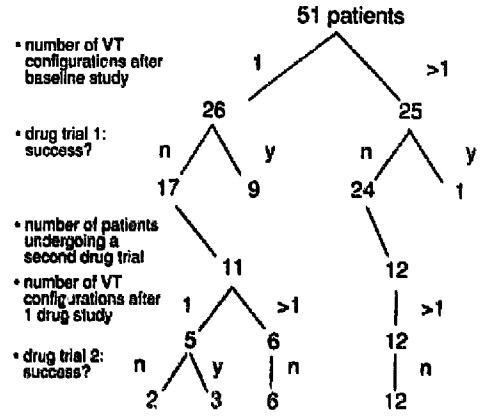


**Figure 4.** Flowchart of outcome of serial electrophysiologic testing. After the first antiarrhythmic drug trial, those patients with one ventricular tachycardia (VT) QRS complex configuration were more likely to have successful electrophysiologic study-guided therapy than were patients with multiple monomorphic QRS complex configurations ($p = 0.016$). Of those patients who had an unsuccessful first antiarrhythmic drug trial and had a second electrophysiologic study-guided antiarrhythmic drug trial, 3 of 5 patients with one ventricular tachycardia QRS complex configuration had successful guided therapy compared with none of 18 patients with multiple ventricular tachycardia QRS complex configurations. n = no (not successful); y = yes (successful). See text for discussion.

underwent a second drug trial. Although 11 of these 23 patients had only one monomorphic ventricular tachycardia QRS complex configuration at baseline, 6 of these 11 patients had a second monomorphic ventricular tachycardia QRS complex configuration induced during the first drug trial. Thus, after the second drug trial, 5 of the 23 patients still had only one monomorphic ventricular tachycardia QRS complex configuration. None of 18 patients who had two monomorphic ventricular tachycardia QRS complex configurations after one drug trial subsequently had a successful second drug trial. However, three of the five patients with a single ventricular tachycardia QRS complex configuration after the first unsuccessful drug trial subsequently had a successful electrophysiologic study-guided drug trial.

**Three or more monomorphic ventricular tachycardia QRS complex configurations.** No patients with three or more monomorphic ventricular tachycardia QRS complex configurations had successful electrophysiologic study-guided therapy. After the baseline study and serial studies, 16% and 41% of patients had three or more ventricular tachycardia configurations, respectively.

**Predictive value.** The negative predictive value was consistently high, ranging from 94% to 97%. The positive predictive value ranged from 36% to 60% (Table 3). The consistently high negative predictive value indicated that the observation of two or more ventricular tachycardia QRS complex configurations predicted failure of serial electrophysiologic study-guided drug testing. Therefore, a second monomorphic QRS complex ventricular tachycardia, whether observed at baseline study during reinduction, at baseline study during attempted pacing termination or at drug trial testing, predicted failure of electrophysiologic study-guided drug therapy.

**Stepwise multiple logistic regression analysis.** We examined several other clinical and electrophysiologic variables

**Figure 3.** Histogram demonstrating outcome of electrophysiologic study-guided antiarrhythmic drug therapy based on the number of ventricular tachycardia (VT) QRS complex configurations observed at baseline study (including ventricular tachycardias resulting from attempted pacing termination). The difference between successful therapy in the two groups (1 vs. ≥2) was statistically significant. Symbols as in Figure 2. See text for discussion.

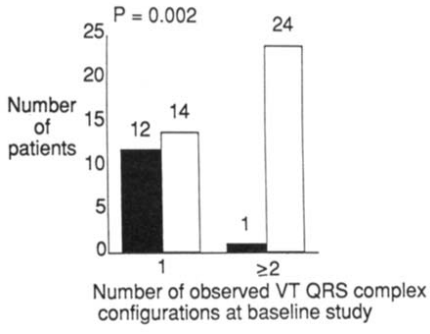


**Table 3.** Positive (+) and Negative (−) Predictive Values of the Number of Monomorphic Ventricular Tachycardia Configurations

| | + | − |
|---|---|---|
| Baseline study | | |
| Induced configurations | 36% | 94% |
| Observed configurations | 46% | 96% |
| Initial study (baseline study and intravenous procainamide trial) | 60% | 97% |

Induced configurations = monomorphic QRS complex ventricular tachycardia configurations that resulted from programmed stimulation induction; observed configurations = monomorphic QRS complex ventricular tachycardia configurations that resulted from programmed stimulation induction or attempted pace termination.

Table 4. Multivariate Stepwise Logistic Regression Analysis

| | p Value |
|---|---|
| Age | NS |
| Gender | NS |
| Left ventricular ejection fraction | 0.03 |
| No. of extrastimuli to induce VT | NS |
| Induced VT cycle length | NS |
| 1 versus ≥2 VT QRS complex configurations | 0.01 |
| Mode of clinical presentation | NS |

VT = ventricular tachycardia.

to determine whether they predicted outcome. The number of monomorphic ventricular tachycardia QRS complex configurations ($p = 0.01$) and left ventricular ejection fraction ($p = 0.03$) demonstrated significance on multivariate logistic stepwise regression analysis (Table 4).

## Discussion

**Prognostic significance of multiple monomorphic ventricular tachycardia configurations during serial electrophysiologic testing.** Previous studies have shown that patients with recurrent ventricular tachycardia often exhibit multiple QRS complex configurations (1,18,19,21–23). Wilber et al. (21) reported that 61% of patients had induced ventricular tachycardias with different QRS complex configurations at electrophysiologic study. Furthermore, Cooper et al. (22) showed that ventricular tachycardias with different QRS complex configurations were reinduced in 36% of patients during one baseline study and in 74% of patients during baseline studies repeated on different days. Our study again demonstrated that monomorphic ventricular tachycardias with multiple QRS complex configurations are commonly induced during electrophysiologic study.

More important, this study demonstrated that initiation of monomorphic ventricular tachycardias with multiple QRS complex configurations at baseline study predicted subsequent failure of electrophysiologic study–guided testing. In our study, multiple monomorphic QRS complex ventricular tachycardias were initiated either by inducing morphologically different tachycardias with programmed ventricular stimulation or by attempted pacing termination of ventricular tachycardia. Furthermore, our results showed that the occurrence of multiple monomorphic QRS complex ventricular tachycardias had a high negative predictive value whether they were observed during induction or attempted pacing termination at the baseline state or during drug testing. Therefore, by counting *all* observed sustained ventricular tachycardias rather than only all induced ventricular tachycardias at baseline study, more patients were found to have multiple QRS complex ventricular tachycardia configurations (49% vs. 36%). Regardless of how and when multiple monomorphic ventricular tachycardia QRS complex configurations were present during the course of serial electrophysiologic studies, our data indicate that their presence indicated a very low likelihood of finding a successful electrophysiologic study–guided drug.

**Predictors of success or failure of serial electrophysiologic studies.** In our study, multiple monomorphic QRS complex ventricular tachycardias and a low left ventricular ejection fraction were independent predictors of failure of serial electrophysiologic drug studies. Past studies have shown low left ventricular ejection fraction to be a useful predictor of this outcome (5,7). The absence of coronary artery disease has been observed to be a predictor of success of serial drug studies (8). Other useful predictors previously cited include the nature of the presenting arrhythmia, induction of ventricular tachycardia with one ventricular extrastimulus and response to intravenous procainamide (5–7).

Although a positive response to intravenous procainamide has been cited as a useful predictor, a negative response to intravenous procainamide has been reported to have limited utility (5). In our study, after one unsuccessful drug trial (oral or intravenous), 3 (60%) of 5 patients who still had only one observed ventricular tachycardia QRS complex configuration subsequently had suppression of ventricular tachycardia induction at the second trial compared with none of 18 patients who had multiple monomorphic ventricular tachycardia configurations observed. Therefore, in our study, a negative response to a first drug trial did not necessarily predict subsequent failure. Rather, it depended on the number (one or two or more) of monomorphic ventricular tachycardia QRS complex configurations observed.

**Limitations of the study.** Because this was a retrospective review, the same rigid electrophysiologic study protocol was not followed in all patients. Only patients who had reproducible induction of sustained monomorphic ventricular tachycardia were included in this study. Therefore, we cannot comment on those patients who did not have reinduction of ventricular tachycardia performed, which generally included patients with inducible, rapid ventricular tachycardia that was either hemodynamically unstable or degenerated to ventricular fibrillation.

Additionally, only 49% of patients received an intravenous trial of procainamide. Had more patients undergone an intravenous antiarrhythmic drug trial, more patients might have demonstrated multiple monomorphic QRS complex ventricular tachycardias. However, lack of consistency of intravenous procainamide testing does not limit the results or conclusions with regard to multiple monomorphic ventricular tachycardia configurations observed at baseline study.

Patients in our study had a mean of 1.5 drug trials. Successful suppression of ventricular tachycardia induction is often achieved in the first one to two drug trials (9). Given that the number of drug trials in our study was relatively small, a bias may have been introduced. The probability of inducing multiple monomorphic QRS complex tachycardias might have increased had more drug trials been performed. However, this bias may be difficult to sort out because

patients and physicians are increasingly unwilling to undergo numerous electrophysiologic trials to assess drug efficacy.

We included in our analysis monomorphic ventricular tachycardia that resulted from attempted pacing termination of an initially induced ventricular tachycardia. Because these ventricular tachycardias may be considered "nonclinical," it remains controversial whether they indeed identify patients with multiple monomorphic ventricular tachycardias. In our series, in all patients with a morphologically distinct monomorphic ventricular tachycardia resulting from antitachycardia pacing, serial drug testing failed. Thus, we believe that the presence of multiple monomorphic ventricular tachycardias, regardless of how the tachycardia was induced, identifies patients with a very low probability of having successful antiarrhythmic drug therapy selected by serial electrophysiologic study–guided drug testing.

**Clinical implications.** Because of the increasing economic pressure on physicians to limit diagnostic tests and hospital days, we suggest that the following strategy may streamline serial electrophysiologic drug testing. At each step during serial electrophysiologic studies, patients can be stratified by the number of monomorphic ventricular tachycardia QRS complex configurations observed. Whenever two or more configurations are observed, the patient should be considered for alternative modes of treatment. However, for those patients who have only one demonstrated ventricular tachycardia QRS complex configuration, serial electrophysiologic study–guided drug testing should be pursued.

**Conclusions.** In patients with coronary artery disease, multiple monomorphic ventricular tachycardia QRS complex configurations during baseline study predicted failure of electrophysiologic study guided drug therapy. The observation of a second monomorphic ventricular tachycardia configuration at subsequent drug trials also predicted failure of serial electrophysiologic drug testing.

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